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The molecular mechanisms of liver and islets of Langerhans toxicity by benzene and its metabolite hydroquinone in vivo and in vitro.

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Abstract

Benzene (C6H6) is one of the most commonly used industrial chemicals causing environmental pollution. This study aimed to examine the effect of benzene and its metabolite hydroquinone on glucose regulating organs, liver and pancreas, and to reveal the involved toxic mechanisms, in rats. In the in vivo part, benzene was dissolved in corn oil and administered through intragastric route at doses of 200, 400 and 800 mg/kg/day, for 4 weeks. And, in the in vitro part, toxic mechanisms responsible for weakening the antioxidant system in islets of Langerhans by hydroquinone at different concentrations (0.25, 0.5 and 1 mM), were revealed. Benzene exposure raised the activity of phosphoenolpyruvate carboxykinase (PEPCK), glucose 6-phosphatase (G6Pase) enzymes and increased fasting blood sugar (FBS) in comparison to control animals. Also, the activity of hepatic glucokinase (GK) was decreased significantly. Along with, a significant increase was observed in hepatic tumor necrosis factor (TNF-α) and plasma insulin in benzene treated rats. Moreover, benzene caused a significant rise in hepatic lipid peroxidation, DNA damage and oxidation of proteins. In islets of Langerhans, hydroquinone was found to decrease the capability of antioxidant system to fight free radicals. Also, the level of death proteases (caspase 3 and caspase 9) was found higher in hydroquinone exposed islets. The current study demonstrated that benzene and hydroquinone causes toxic effects on liver and pancreatic islets by causing oxidative impairment.

KEYWORDS: Benzene; environmental pollutants; glucose homeostasis; hydroquinone; oxidative stress

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